LEVEL II (13)

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A LITERATURE REVIEW - PROBLEM DEFINITION STUDIES ON SELECTED TOXIC CHEMICALS

ADA058508

AD No. _____
DDC FILE COPY

Contract Summary Report

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June 1978

Supported by

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
FORT DETRICK, FREDERICK, MARYLAND 21701

Contract No. DAMD-17-77-C-7020

DDC
RECEIVED
SEP 13 1978
F

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SCIENCE INFORMATION SERVICES DEPARTMENT
THE FRANKLIN INSTITUTE RESEARCH LABORATORIES
THE BENJAMIN FRANKLIN PARKWAY · PHILADELPHIA, PENNSYLVANIA 19103

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER

2. GOVT ACCESSION NO.

3. RECIPIENT'S CATALOG NUMBER

4. TITLE *(and Subtitle)*

(16) A Literature Review - Problem Definition Studies on Selected Toxic Chemicals. Contract Summary Report,

5. TYPE OF REPORT & PERIOD COVERED

(9) Contract Summary Report, March 1977 - April 1978,

6. PERFORMING ORG. REPORT NUMBER

7. AUTHOR(s)

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Principal Investigator
P.N. Craig, Ph.D. and
Jon E. Villaume, Ph.D.

8. CONTRACT OR GRANT NUMBER(s)

(15) DAMD-17-77-C-7020

9. PERFORMING ORGANIZATION NAME AND ADDRESS

Science Information Department
The Franklin Institute Research Laboratories
The Benjamin Franklin Parkway - Phila. PA. 19103

10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS

62720A
(No) 3E762720A835 00.030
(17) 00

11. CONTROLLING OFFICE NAME AND ADDRESS

U.S. Army Medical Research and Development Command
Fort Detrick, Frederick, Maryland 21701

12. REPORT DATE

June 16, 1978

13. NUMBER OF PAGES

83

14. MONITORING AGENCY NAME & ADDRESS *(if different from Controlling Office)*

15. SECURITY CLASS. *(of this report)*

Unclassified

15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT *(of this Report)*

Approved for Public Release; Distribution Unlimited

(11) 16 Jun 78 (12) 83p.

17. DISTRIBUTION STATEMENT *(of the abstract entered in Block 20, if different from Report)*

(10) Khizar /Wasti, Paul N. /Craig
Jon E. /Villaume

18. SUPPLEMENTARY NOTES

19. KEY WORDS *(Continue on reverse side if necessary and identify by block number)*

Animal toxicology	Environmental fate	Industrial hygiene
Aquatic toxicity	Environmental impact	Pharmacokinetics
Cetyl alcohol	Fog oils	Phosphorus
Diesel fuel	Human toxicology	Physical & chemical properties

20. ABSTRACT *(Continue on reverse side if necessary and identify by block number)*

Problem Definition Studies (PDS) on potentially Toxic Chemicals were prepared. This report gives an overview of the efforts involved in the preparation of the PDS and the subject areas covered in these studies.

78 14 08 276

#10 414

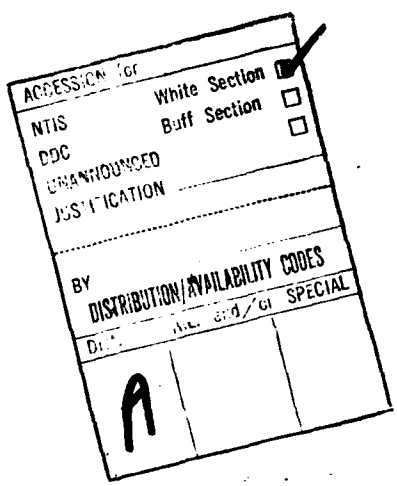
DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

19. Key Words (continued)

Phytotoxocity
Problem Definition Studies
Resins
Safety aspects
SGF Nos. 1 & 2
Smoke
TNT
Urea-formaldehyde
Zinc chloride



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ABSTRACT

Problem Definition Studies (PDS) on potentially Toxic Chemicals were prepared. This report gives an overview of the efforts involved in the preparation of the PDS and the subject areas covered in these studies.

CONTRACT SUMMARY REPORT

The Franklin Institute Research Laboratories prepared Problem Definition Studies (PDS) on potentially Toxic Chemicals under the referenced contract.The following eight Problem Definition Studies were prepared.

1. - Occupational Health and Safety Aspects of Diesel Fuel and White Smoke generated from it (Volume 1).

2. - Occupational Health and Safety Aspects of Phosphorus Smoke Compounds (Volume 2).

3. - Occupational Health and Safety Aspects of 2,4,6- Trinitrotoluene (TNT) (Volume 3).

4. - Occupational Health and Safety Aspects of the Fog Oils, SGF No. 1 and SGF No. 2 and Smoke Screens generated from them (Volume 4).

5. - Occupational Health and Safety and Environmental Aspects of Zinc Chloride (Volume 5).

6. - Occupational Health and Safety and Environmental Aspects of Urea-Formaldehyde Resins (Volume 6).

7. - Occupational Health and Safety and Environmental Aspects of Cetyl Alcohol (Volume 7).

8. - Environmental Aspects of Diesel Fuels and Fog Oils SGF No. 1 and SGF No. 2 and Smoke Screens generated from them (Volume 8).

The PDS Volume 1 - 4 cover the following subject areas:

1. - Physical and Chemical Properties

2. - Human toxicology including epidemiology

3. - Animal toxicology

4. - Pharmacokinetics

5. - Industrial hygiene and safety practices and standards

6. - Sampling and analysis

7. - Recommendations for further research

The PDS Volume 5 - 7 contain all the above mentioned subjects and in addition cover environmental aspects of the respective chemicals including the following topics:

1. - Occurrence, dispersion, and fate in the environment

2. - Aquatic toxicity

3. - Phytotoxicity

4. - Effects on domestic animals and wildlife

5. - Environmental impact

The PDS Volume 8 covered the environmental aspects of diesel fuel and fog oils, SGF No. 1 and SGF No. 2. The table of contents of all the PDS are included in Appendix 1. The executive summaries of the eight PDS are included in Appendix 2.

The literature search performed for the preparation of the PDS included both manual and the computerized sources. In addition, several books, encyclopedias, and review articles were consulted. Personal contacts were made in order to obtain unpublished relevant information. The complete lists of sources of the literature search and the personnel contacted for each PDS are included in Appendix 3. Copies of each problem definition study have been distributed according to the list provided by USAMRDC. The hard copies of the references cited in the problem definition studies have been provided to the USAMBRDL, Fort Detrick.

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APPENDIX 2

Diesel Fuel (Volume 1)

EXECUTIVE SUMMARY

This report is a literature review (75 references) which covers physical and chemical properties, analytical methods, experimental animal studies and occupational health and safety of diesel fuel and the white smoke generated from it.

Diesel fuel is a refined, distilled petroleum product made from crude oil. The chemical composition of diesel fuel varies from batch to batch, but the fuel is required to have certain other properties which have been specified by the U.S. Armed Forces. Certain additives may be added to improve engine performance. The smoke which is produced from diesel fuel consists of tiny oil droplets suspended in air in a concentration great enough to produce a dense white cloud.

Inhalation of diesel oil smoke may cause lung damage, although further studies are needed. Irritation of the skin occurs in some individuals who handle diesel fuel in industry. The skin irritation consists of dryness, redness and pimples, sometimes severe enough to leave scars. Whether it is due to an allergy or to carelessness and uncleanliness is uncertain. Swallowing diesel fuel may severely damage the stomach lining. If diesel fuel is taken into the lungs, severe irritation, coughing, and inability to breathe may result. The lung condition may clear up or death may occur, especially in accidents when children drink and choke on diesel fuel. The possible harmful effects to humans during long-term exposure to diesel fuel white smoke have not been studied.

In studies on experimental animals, diesel fuel white smoke produced reduced ability to fight infection in rats, as well as loss of coordination, tiredness, and dry flaking skin. Rats fed diesel fuel developed abnormal blood, liver damage, and some died. When rat skin was painted with diesel fuel, pimples, hair loss and peeling of skin occurred. Some diesel fuel batches caused skin cancer after painting the fuel repeatedly on mouse skin. No studies are reported for effects of diesel fuel on reproduction in animals.

When taken into the body, some of the aromatic compounds of diesel fuel are metabolized in the liver. Arene oxides, formed as intermediates in the metabolism, may be cancer producing or cause mutations. Diesel fuel may be excreted from the body in the urine, or exhaled from the lungs.

Persons who may be exposed to diesel fuel white smoke should be protected from breathing the smoke and getting it in eyes, on skin and hair. Medical examinations should be provided to check for lung problems, skin problems or other disturbances in persons exposed to the smoke.

Phosphorus Smoke Compounds (Volume 2)

EXECUTIVE SUMMARY

This literature review (100 references) discusses the occupational health and safety of phosphorus smoke compounds which are used by the U.S. Armed Forces to produce smoke screens. These five compounds are white phosphorus, red phosphorus, butyl rubber/red phosphorus, butyl rubber/white phosphorus, and epoxy white phosphorus.

To make butyl rubber/red phosphorus, red phosphorus is combined with styrene-butadiene rubber (butyl rubber). White phosphorus also combines with styrene-butadiene rubber to make butyl rubber/white phosphorus, which is also called plasticized white phosphorus. Epoxy white phosphorus is made from white phosphorus and Bisphenol A-epichlorohydrin, an epoxy resin.

There are no acceptable reports on the dangers of red phosphorus to the health of humans or animals.

White phosphorus causes severe skin burns upon contact with the skin. Breathing white phosphorus vapors can damage the lungs and liver. If white phosphorus is eaten (children may eat it accidently in roach poison) it may cause garlic taste, vomiting and damage to the stomach and the rest of the digestive tract. Death may occur in 12 hours. If the person lives, after a few days the patient may sicken and begin vomiting again, and this means that there may be damage to the kidneys, liver and digestive tract.

When white phosphorus fumes are inhaled repeatedly, in industry, damage to the jawbone, teeth, and other bones is reported. Also there may be blood disorders and abnormal urine.

In animals, eating of white phosphorus causes liver damage and death. Placing it on skin of rabbits caused deaths to many of them in 3 days. It did not produce eye irritation or skin allergy in rabbits. Liver, kidney and blood changes occurred. Long-term animal studies with white phosphorus have caused retarded growth and damage to bones, liver, kidneys and brain. The ability of white phosphorus to produce cancer in animals, mutations in bacteria, or to harm unborn offspring is not known.

When smoke screens are made from burning phosphorus smoke compounds, humans who breathe the smoke may begin coughing, and develop sore throats, runny noses and may have trouble breathing after 15 minutes of breathing the smoke. They recover in a few days if they stop breathing smoke. In animal experiments, breathing the same smoke caused lung congestion, and liver and kidney damage.

Mice which breathed burning styrene-butadiene rubber died of asphyxiation from carbon monoxide. There are no experiments reported to test the hazards of breathing butyl rubber/phosphorus smoke screens, or the epoxy white phosphorus smoke screen. Also, there is no information on the ability of the butyl rubber/phosphorus or epoxy white phosphorus smoke to produce cancer, mutations or to harm unborn offspring.

In industries where persons work around white phosphorus, protection from burns to the eyes and skin is important. Gas masks are necessary to avoid breathing large amounts of white phosphorus fumes. White phosphorus is also a fire hazard because it will burn if it is exposed to room air. The fire can be extinguished with sand or water. Persons working with white phosphorus should be examined routinely for damage to the teeth and jawbone, and have blood and urine tests done.

TNT (Volume 3)
EXECUTIVE SUMMARY

This literature review (224 references) discusses various topics related to occupational health and safety of TNT, an explosive used in bombs, shells and grenades. It is a yellow powder and crystals at room temperature, and can be stored at room temperature for 20 years without decomposing.

Manufacture of TNT creates harmful fumes of TNT and other gases. Breathing the fumes can cause sore throat, sneezing or choking, and lung damage may occur, with recovery after several days. Workers who are exposed to TNT by breathing fumes or getting TNT on the skin may experience harmful effects including liver malfunction, decreased ability of the bone marrow to make blood cells, and death. TNT may also damage the heart, blood vessels, kidneys, pancreas, and possibly cause cataracts. Skin rash and redness may develop on the hands and forearms, collar area and ankles. Nausea and vomiting and stomach pains also are reported. The color of urine of persons with TNT poisoning is darker than normal, and may be red, but not bloody. Removal of the worker from all TNT fumes is important in treatment of TNT poisoning. Deaths are usually due to liver or blood disease. OSHA requires that workers in TNT plants are not exposed to more than 1.5 mg/m^3 of TNT in air, but disease still appears in some workers. The U.S. Army has lowered its acceptable TNT level to half of this amount, 0.5 mg/m^3 of TNT, in order to provide greater protection to exposed workers.

In animals, cats are more sensitive to TNT toxicity than rabbits, rats and monkeys. Liver disease and blood disease appear in animals exposed to TNT fumes. Dogs fed TNT showed no disease other than vomiting. Dogs which inhaled TNT fumes lost weight, and had vomiting and diarrhea and some blood problems but no liver disease. TNT cancer has not been reported in laboratory animals or humans exposed to TNT. But TNT did cause mutations in bacteria and chromosomal abnormalities in bone marrow cells of rats. The effect of TNT on reproduction is not known.

TNT can be absorbed into the body through skin, but much less enters the body by breathing fumes than through the skin. When fumes enter the mouth there is a bitter taste. The liver breaks down TNT which enters the body, and these breakdown products of TNT are found in urine but not in feces.

Bacteria also can degrade TNT in waste water. Sunlight causes TNT to decompose in water, and gives the water a pink or red color. Organisms living in water can be harmed by TNT waste which is disposed in the water.

Persons working in TNT plants should not be exposed to TNT in air in concentrations above the OSHA limit. They should get medical examinations routinely and tests for blood and liver function, in order to detect signs of TNT disease early enough to cure the worker. Clean work clothes and showers after work may help avoid TNT skin contamination, and safety glasses, face shields and other protection are important.

The effects of TNT on reproduction and on unborn offspring, cancer from TNT, and eye cataract from TNT, are some topics which need to be studied.

Fog Oils (Volume 4)

EXECUTIVE SUMMARY

This literature review (144 references) discusses the problems related to fog oil exposure of humans and other mammals including rats, mice, guinea pigs, dogs, cats, hamsters, livestock and rhesus monkeys. There are two fog oils: SGF No. 1 and SGF No. 2. SGF No. 1 fog oil is a fuel oil distilled from crude petroleum, very similar to home heating oil, diesel fuel and some kerosenes. SGF No. 2 fog oil is a lubricating oil originating from crude petroleum, very similar to S.A.E. 10 motor oil and some industrially used lubricants and cutting oils. Its yellow color indicates that it is less refined than white mineral oil, a once commonly used laxative. Both fog oils can be used in smoke pots and mechanical smoke generators which produce dense white smoke screens. The smoke is actually made up of microdroplets of fog oil.

Army personnel who worked under fog oil smoke screens in World War II may be examined to learn whether or not there are long-term health hazards from fog oil smoke. In industry, workers exposed to lubricating oil mists frequently have skin conditions such as acne. Skin cancer also has been reported in these workers, as well as other cancers in the lung and throat. On the other hand, workers exposed on the job to heating or fuel oil mists usually complain only of skin irritations.

The inhalation of oil mists of SGF No. 1 fog oil caused lung tumors in mice and stomach tumors in monkeys. The oil accumulated in the lungs, but did not usually cause pneumonia. When exposed to oils similar to SGF No. 1, animals have developed pneumonias, adverse effects on the blood and bone marrow, skin and eye irritation and hair loss, and death at high doses.

Laboratory animals exposed to lubricating oils like SGF No. 2 have developed skin and lung tumors. Breathing of oil mists caused oil to accumulate in the lungs. In addition, pneumonia, adverse effects on the liver, spleen, kidneys, colon, skin and heart have sometimes resulted.

Although information on the ways in which the body absorbs, metabolizes, stores and excretes the fog oils is incomplete, it is established that mutagenic substances are formed in the enzymatic breakdown of some aromatic petroleum hydrocarbons found in SGF No. 2 type oils.

Recommendations for the further study of adverse effects of fog oils in occupationally exposed humans and in laboratory animals include evaluations of carcinogenicity and other long-term effects. There is a need to measure the air concentrations of fog oil smoke produced from smoke generators or smoke pots during present-day military smoke screening operations. It is also recommended that individuals who are exposed to the fog oil smoke screens have protection against breathing the smoke and against wearing oily clothing next to the skin or scalp.

Methods of collection of fog oil smoke in the air and measuring the air concentration of fog oil are presented. In animal experiments, it is useful to measure the accumulation of oil in the lungs and other parts of the body

after fog oil exposures. Appropriate methods to make this oil more visible and to measure its accumulation in various organs are included.

The environmental impact of using fog oil smoke screens is presented as a separate document included in this series of reports.

Zinc Chloride (Volume 5)

EXECUTIVE SUMMARY

Zinc chloride is a white, odorless compound which readily dissolves in water. It has been used by the military as a smoke screen to protect personnel, in metal and textile industries, and for fire-fighting exercises among others.

Zinc chloride smoke is a potential health hazard, especially when generated in an enclosed space with inadequate ventilation. Persons breathing in high concentrations suffer severe pulmonary irritation. The lungs can become filled with fluid and the lung tissue may be destroyed. Extended exposure to high concentrations can be fatal. Skin contact with aqueous zinc chloride solutions causes severe burns especially if contact occurs around a pre-existing wound. Oral intake of zinc chloride paste by a child was reported to produce corrosive gastritis and liver necrosis, and proved fatal. Eye and nose contamination with zinc chloride by 2 workers caused burns on the eyes, permanently impaired vision, and permanent loss of the sense of smell.

Laboratory dogs exposed to high concentrations of zinc chloride smoke developed fluid in their lungs. Application of a solution of zinc chloride to the shaved skin of guinea pigs slowed growth but did not cause deaths. However, an injection of a zinc chloride solution into the abdomens of guinea pigs caused the deaths of 8 out of 10 animals in one week; 6 died within 24 hours. Prolonged oral intake of a solution of zinc chloride in addition to a diet deficient in pantothenic acid, caused vitamin deficiency symptoms and retarded growth in rats. In another study, prolonged oral intake of zinc chloride did not affect reproduction and normal young were born. Rats given intraperitoneal injections of zinc chloride for several weeks developed kidney and nerve cell injuries and abnormalities.

There have been no reported cases of carcinogenicity due to zinc chloride exposure in humans. Except for the ability to induce tumors by intratesticular injections in fowl, no evidence exists that zinc chloride is carcinogenic in animals by oral or intraperitoneal routes of administration.

No evidence exists in the literature that zinc chloride is mutagenic. Zinc chloride has produced teratogenic effects when injected into the yolk sacs of chicken eggs.

Zinc chloride is toxic to freshwater and marine organisms. The toxic concentrations of zinc chloride to fishes vary with species and water conditions. However, concentrations as low as 0.17 mg/l of zinc chloride in water have been found to be lethal. In the embryos of clams, sand dollars, and sea urchins, zinc chloride induced mortalities and abnormal development.

Exposure to zinc chloride and gamma radiation, alone or in combination, decreased the survival of a strain of bacteria. Immersion of corn and tomato leaves and cauliflower, lettuce, and carrot cultures into zinc chloride solutions caused leaf injury and inhibited growth. Zinc is required for normal plant growth but excess concentrations can accumulate and have toxic effects.

Zinc is absorbed through the skin and gastrointestinal tract as evidenced by experiments with radioactive zinc chloride in man and animals. Absorbed zinc is distributed in the tissues with the highest concentrations being found in the liver. The primary route of excretion of zinc was through the feces. In experiments with aquatic organisms, radioactive $zinc^{65}$ was distributed in the tissues of clams, mussels, and sea urchins.

Zinc can accumulate in plants, aquatic organisms, domestic animals and wildlife which are consumed by humans. These represent a possible route for the accumulation of zinc in the food chain. Zinc occurs naturally in rocks, water, plants, animals and man. It is dispersed in the environment by zinc chloride smoke screens and by various industries. Several factors, such as soil condition and climate determine the subsequent movement of zinc and its fate in the environment.

Zinc chloride is produced as a smoke from HC (hexachloroethane) smoke pots. HC smoke mix is a solid mixture of grained aluminum, zinc oxide, and hexachloroethane in percentages of 6.68, 46.66, and 46.66, respectively. The environmental impact of this zinc chloride cloud and resultant fallout are subsequently reviewed.

Urea-formaldehyde (Volume 6)

EXECUTIVE SUMMARY

The literature review (88 references) discusses the occupational health and safety of urea-formaldehyde resins.

Urea-formaldehyde resins are thermosetting plastics used extensively in adhesive, molding, laminating, coating, insulation, textile and paper treatment. These resins are a mixture of condensation products of urea and formaldehyde. The composition and structure of these products depend on: a) conditions of the reaction, b) degree of polymerization, and c) molar ratio of the two components. The foam form of the resin is prepared by the interaction of urea and formaldehyde in the presence of a catalyst and a surfactant.

Low molecular weight products exist either as a water-soluble viscous liquid or a white powdery solid. These are converted by heat or acid into high polymeric forms which are water-insoluble amorphous solids. These two groups of resins will be discussed separately.

The high polymeric forms of the resins are colorless, transparent, odorless and tasteless solids, insoluble in cold water, cold dilute acids or alkalis. They are not affected by common organic solvents, oil or grease, but are decomposed in boiling water or in hot strong acids or alkalis. No studies on their effects on birds, aquatic organisms or wildlife are found in the literature. When the resins are ingested by animals, they are excreted in the feces without being metabolized. Since they are very stable under ambient conditions, accumulation in the environment is expected.

The low molecular weight condensation products, on the other hand, have quite different properties and effects. They are soluble in water and alcohol and decompose easily with the release of formaldehyde, which is the main cause of toxic effects.

Formaldehyde is highly irritating to eyes, skin, and mucous membrane. It is generated (1) during manufacture of the resins because it is one of the components, (2) during processing of the resins when heat is applied, such as molding, laminating, etc., (3) from resin products which contain incompletely reacted formaldehyde, and (4) from finished products which are not completely polymerized and decompose slowly to release formaldehyde.

Toxicity studies on urea-formaldehyde resins have focused upon the effects of formaldehyde which is generated as discussed above. Eye irritation was reported among factory workers and store employees where resin-treated fabrics were sold. Dermatoses developed among persons who came into direct contact with the resins, such as workers engaged in processing the resins and individuals wearing clothing made from resin-treated fabrics, which contain insufficiently polymerized resin.

Cetyl Alcohol (Volume 7)

EXECUTIVE SUMMARY

Cetyl alcohol, a white, crystalline, waxlike solid with a rose odor, is insoluble in water but soluble in several organic solvents. It is used in a wide variety of industrial and consumer products such as cosmetics and pharmaceuticals, as an evaporation suppressant on water reservoirs and soils, and as an antitranspirant on plants.

Contact with cetyl alcohol-treated tent canvas and subsequent application of skin creams containing an unspecified concentration of cetyl alcohol caused oozing dermatitis and swelling of the skin in a 28-year-old woman. No other human toxicity studies were located.

Inhalation of 2220 mg/m^3 synthetic cetyl alcohol for 6 hours resulted in the death of all exposed rats. Oral administration of 1% cetyl alcohol in charcoal to rats produced a cathartic effect. Ingestion of 2 g/day cetyl butyrate or 12 g/day of a diet containing 20% cetyl oleate, esters of cetyl alcohol, induced hyperemia and edema of the intestinal tract, and seborrhea in rats while administration of 2 g/day cetyl oleate plus tributyrin (glyceryl tributyrate) induced only hyperemia and edema of the intestinal tract. Oral administration of 0.25 mg/day cetyl alcohol for 4 months in mice maintained on a vitamin E deficient diet postponed the onset of the effects of the vitamin deficiency. Cutaneous application of an unspecified dose of cetyl alcohol on the exposed skin of guinea pigs caused slight irritation.

Cutaneous application of cetyl alcohol to the skin of mice pretreated with dimethyl benzanthracene (DMBA) caused local irritation. One papilloma was observed in one animal treated with cetyl alcohol and DMBA. The development of adenomas, papillomas, and carcinomas was reported following bladder implantation of an unspecified dose of cetyl alcohol in mice. However, the tumor-promoting ability of cetyl alcohol is inconclusive since similar results were observed in controls following bladder implantation of cholesterol pellets.

Cetyl alcohol is absorbed through the intestinal mucosa of mammals. The alcohol is partly metabolized to palmitic acid and subsequently incorporated into glycerides, phospholipids, and cholesteral esters. Following oral administration of [1-^{14}C]cetyl alcohol, the absorbed activity was primarily recovered in the lymph lipids. Cetyl alcohol is excreted in the urine as conjugated glucuronic acids, unchanged in the feces, or as expired carbon dioxide. Fish incorporate dietary cetyl alcohol as the alcohol and palmitic acid into roe wax esters. The alcohol undergoes oxidation to the corresponding fatty acid and is then reduced again for incorporation into roe wax esters.

Rapid but transitory increases of several freshwater bacterial populations were caused by the application of 17,115 or 70,000 kg of cetyl alcohol to reservoirs as an evaporation suppressant. The bacteria, however, degraded the cetyl alcohol monolayer and, therefore, the density of the populations could not be maintained.

Algal populations of reservoirs increased following application (17,115 or 70,000 kg) of cetyl alcohol and the growth of sedges along the shoreline was also enhanced. Soil and foliar application of cetyl alcohol as an antitranspirant depressed growth and photosynthesis in plants, caused leaf deformities, and, in some cases, caused the ultimate death of treated tissue.

Cetyl alcohol monolayers on reservoirs or other bodies of water may prevent the emergence of some insects and could have an adverse effect on their populations. No mortalities were reported for minnows exposed to 1, 10, 100, or 500 mg/l of cetyl alcohol for 5 days. However, dissolved oxygen depletion and a rise in temperature due to evaporation reduction may result in stress to sensitive fish species (species not specified).

Cetyl alcohol occurs naturally as the cetyl ester of palmitic acid in sperm oil from the head and vertebrae of the sperm whale and is a major component of fish roe wax esters. The alcohol is dispersed into the environment by the industries which utilize or manufacture it and by the use of evaporation suppressants and antitranspirants. Cetyl alcohol can be transported through soil by several means. In the environment, cetyl alcohol is degraded by microorganisms (*Pseudomonas, Coryneform Alcaligenes*, etc.) found in soil and water. It is oxidized to carbon dioxide. Another degradation have not been identified.

Diesel Fuel and Fog Oils (Volume 8)

EXECUTIVE SUMMARY

In this report, studies are reviewed on topics related to the environmental damage caused by diesel fuel and fog oils. The oils are used in U.S. Army smoke screening operations. Both of these petroleum oils, in sufficient quantity, are toxic to ducks, freshwater and marine fish, clams, shrimps and other shellfish, algae, soil and water microorganisms, soil and marine worms, and insects. They cause harmful effects in trees and plants, such as yellowing of leaves, dwarfing, and interference with photosynthesis and fruit production. Other effects in animals include contamination of feathers of ducks, egg abnormalities in quail, equilibrium disturbances in fish, cancer in soft shell clams and interference with food finding behavior in snails and oceanic bacteria.

Oil smoke consists of tiny droplets of fog oil, which are carried as far as 10 km by air currents before falling back to earth. As the smoke droplets settle, an oil film forms over soil, bodies of water and plants. Oil seeps into soil, where it remains to be slowly biotransformed by bacteria. Rainfall runoff may carry some oil into drinking water supplies and other waters. Oil forms a film when it falls on water, and some of the oil may dissolve in the water, be photolyzed by sunlight, metabolized by microorganisms in the water and bioconcentrated by organisms living in the water. In this way the oil can bioaccumulate through secondary consumption, reaching humans eating fish taken from the contaminated water. The amounts of fog oil or diesel fuel which contaminate the ground, plants and waters in smoke screening areas need to be measured in order to present a more definitive evaluation of the environmental impact of U.S. Army smoke screening operations. It may be possible to expect fog oil to persist in soil, harm vegetation and pollute bodies of water sufficiently enough to introduce the oils into the food chain and possibly harm aquatic organisms feeding or living in smoke screening areas.

APPENDIX 3

Diesel Fuel (Volume 1)

INFORMATION SOURCES EXAMINED

Computer Searchable Data Bases

1. National Technical Information Services - covering 1964 to present (searched on 4/4/77)
2. TOXLINE/TOXBACK (searched on 3/29/77)
3. Chemical Condensates - covering 1972 to present (searched on 4/1/77)
4. BIOSIS Previews - covering 1972 to present (searched on 4/8/77)
5. ISI SCISEARCH - covering 1974 to present (searched on 4/8/77)
6. CANCERLINE (searched on 5/5/77)
7. NIOSH Technical Information Center file - (received on May 15, 77)
8. American Petroleum Institute file - (received on May 15, 77)
9. Defense Documentation Center - (received on May 15, 77)

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2. Index Medicus - V.1 (1927) - V.18 (No. 4), 1977.
3. Excerpta Medica - sections entitled *Toxicology and Pharmacology, Occupational Health and Industrial Medicine, Cancer, Environmental Health and Pollution Control* (covering Vol. 1 through last volume available in 1976) were examined.
4. Engineering Index - (covering 1940 through 1977, issue #3).
5. Biological Abstracts - [covering Vol. 1 (1927) through Vol. 61 1976)].

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Phosphorus Smoke Compounds (Volume 2)

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TNT (Volume 3)

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Zinc Chloride (Volume 5)

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Urea-formaldehyde (Volume 6)

Information Sources Examined

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Cetyl Alcohol (Volume 7)

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Diesel Fuel and Fog Oils (Volume 8)

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